

BLAUTIA-DRIVEN SPERMIDINE SIGNALING REPROGRAMS BREG FUNCTION TO REPAIR GUT IMMUNITY IN ULCERATIVE COLITIS

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Poster

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Live *Blautia coccoides* ameliorated colitis in mice through spermidine production that activated STAT3 in regulatory B cells, with ex vivo confirmation in refractory ulcerative colitis patient samples.

KEY POINTS

- Live *Blautia* treatment significantly improved clinical and histological outcomes in DSS-induced colitis mice, including reduced mucosal ulceration, decreased inflammatory infiltration, and restored tight junction proteins, while heat-killed bacteria provided no benefit.
- The therapeutic effect required bacterial spermidine synthesis, as a spermidine synthesis-deficient *Blautia* mutant failed to restore barrier integrity or expand IL-10-producing regulatory B cells with STAT3 phosphorylation.
- Cytokine profiling showed decreased TNF- α and IL-6 with elevated IL-10 in colonic tissues, and spatial transcriptomics of UC patient biopsies identified niches where *Blautia* co-localized with STAT3-activated regulatory B cells and increased IL-10 transcripts.
- Ex vivo stimulation of PBMCs from anti-TNF-refractory UC patients with *Blautia* metabolites promoted regulatory B cell differentiation and restored suppression of TNF- α release.
- The authors conclude the findings support development of therapies aimed at replenishing spermidine-producing *Blautia* in refractory UC patients.

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